Title: THORACIC EPIDURAL BLOCKADE-INDUCED PULMONARY SYMPATHECTOMY IMPROVES PO_2 IN AN OLEIC-ACID INDUCED MODEL OF PULMONARY EDEMA

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Introduction. Previous studies have suggested that sympathetic nerve involvement may mediate the development of acute pulmonary injury, particularly pulmonary edema.[1] This study utilized an oleic acid-induced model of pulmonary edema in anesthetized sheep[2] to study the impact of pulmonary sympathectomy mediated by thoracic epidural blockade on pulmonary and cardiovascular functions.

Materials and Methods. Sheep (n = 14) were anesthetized with thiamylal (15 mg/kg), tracheally intubated, and mechanically ventilated (F_IO_2, 1.0; tidal volume, 12 ml/kg). Ventilator rate was adjusted to maintain a $PaCO_2$ of 35-45 mm Hg. Anesthesia was maintained with thiamylal infusion at 2-3 $mg \cdot kg^{-1} \cdot min^{-1}$. Blood gas analysis was performed on an IL-1303 Analyzer. Oxygen saturation was measured directly on an IL-282 analyzer. While sheep were prone, the epidural space was punctured via a surgically exposed incision at the level of T3-T4. A 17-ga Tuohy needle was utilized with the hanging drop technique; 2% lidocaine, 0.15 ml/kg, was injected, and, after that, half that volume was injected every hour. Also, acridine red, 10%, 0.06 ml, was injected. Spread of dye, which has the same physical characteristics as lidocaine, was used to confirm the blockade and was determined by autopsy. A 4-lumen, balloon-tip pulmonary artery catheter was placed percutaneously via the external jugular vein and enabled measurement of pulmonary artery pressure, pulmonary capillary wedge pressure, and central venous pressure and calculation of cardiac output. A 5-Fr, thermistor-tip catheter was placed percutaneously in the femoral artery and was used to measure extravascular lung water via the thermal dye technique (Edwards Corp., Santa Ana, CA). The contralateral femoral artery was simultaneously cannulated with a 20-ga catheter to permit continuous recording of arterial blood pressure and sampling of blood. Mean arterial pressure, extravascular lung water, arterial oxygen delivery, cardiac index, venous admixture ($\dot{Q}sp/\dot{Q}t$), PaO_2, $PaCO_2$, and pH were also measured or calculated. Functional residual capacity was measured by the helium dilution technique. After initial baseline measurements, oleic acid, 0.035 ml/kg, was instilled into the main pulmonary artery via the pulmonary artery catheter. After 90 min, lidocaine was (n = 6) or was not (n = 8) administered as part of thoracic epidural blockade, as described above. Data were collected at 90, 120, 180, 240, and 300 min after the instillation of oleic acid. Statistical analysis was by ANOVA and Duncan's multiple range test, $P < 0.05$ being considered significant.

Results. With lidocaine, PaO_2 was significantly higher than that in the control group at 240 and 300 min after injection (table). This was coupled with close to significant differences in $\dot{Q}sp/\dot{Q}t$ (table). At 300 min, O_2 delivery differed significantly with lidocaine. None of the other parameters, measured or calculated, were statistically significantly affected at any of the intervals measured.

Discussion. This study demonstrates that, in animals with oleic acid-induced pulmonary injury, administration of thoracic epidural blockade can minimize the deterioration in oxygenation that normally takes place. Sympathetic blockade appears to influence ventilation-perfusion relationships favorably. That extravascular lung water did not change may indicate that thoracic epidural blockade did not improve lung water clearance. The delay before any observed differences occurred could be attributed to the delay in the onset of sympathetic blockade via the epidural route. In a model of oleic acid-induced pulmonary edema, thoracic epidural blockade with lidocaine appears to ameliorate the changes in oxygenation normally observed.

References

1. Dauber IM, Weil JV: Lung injury edema in dogs. Influence of sympathetic ablation. J Clin Invest 72:1977-1986, 1983
2. Julien M, Hoeffel JM, Flick MR: Oleic acid lung injury in sheep. J Appl Physiol 60:433-440, 1986

TABLE. Effects of Epidural Lidocaine on Oleic Acid - Induced Pulmonary Injury

Time after Oleic Acid Instillation	Control (n = 8)	Lidocaine (n = 6)	P
	Cardiac index		
Baseline	3.6 ± 1.5	3.6 ± 1.0	--
240 min	3.6 ± 8	4.2 ± 1.7	--
300 min	3.3 ± 1.1	4.3 ± 1.8	--
	PaO_2 (mm Hg)		
Baseline	496 ± 54	531 ± 51	--
240 min	59 ± 16	85 ± 27	< 0.05
300 min	50 ± 17	81 ± 33	< 0.05
	Venous adixture(%)		
Baseline	11.6 ± 3.5	10 ± 7	--
240 min	51 ± 12	44 ± 3	< 0.07
300 min	61 ± 1	49 ± 5	< 0.07

Values are means ± SD.